Technical Resources

Citations

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1. Le'Negrate, G., Rotagno, R., Auberger, P., Rossi, B., Hofman, P. (2003) Down regulation of caspases and Fas ligand expression, and increased lifespan of neutrophils after transmigration across intestinal epithelium *Cell Death and Differentiation* **10**, 153-162.

   Notes: Anti-ACTIVE® JNK pAb was used in immunoblot analysis of human polymorphonuclear leukocyte protein lysates. (0002665)

2. Ciesielski-Treska, J., Ulrich, G., Chasserot-Golaz, S., Zwiller, J., Revel, M.O., Aunis, D., and Bader M.F. (2001) Mechanisms underlying neuronal death induced by chromogranin A-activated microglia. *Mechanisms underlying neuronal death induced by chromogranin A-activated microglia.* **276**, 13113-13120.

   Notes: The neurotoxic effects of chromogranin A activated microglia in neurodegenerative diseases was examined. Rat neurons were grown in the presence or absence of conditioned media from chromogranin A treated or untreated microglia. DNA fragmentation in apoptotic neurons was detected using the DeadEnd™ Fluorometric TUNEL System. The levels of dually phosphorylated, active JNK in these cells was determined by Western blot analysis using the Anti-ACTIVE® JNK pAb. A pan JNK antibody was used to normalize for total JNK protein on the Western blots. (0002378)

3. Rumora, L., Shaver, A., Zanic-Grubisic, T., and Maysinger, D. (2001) Differential regulation of JNK activation and MKP-1 expression by peroxovanadium complexes. *Neurochem. Int.* **38**, 341-347.

   Notes: The effect of bisperoxovanadium complexes, known protein tyrosine phosphatase inhibitors, on the activation of JNK was examined in a number of cell lines, including HeLa, PC12 and OVCAR-3. The level of dually phosphorylated (activated) JNK was determined by Western blot analysis using a 1:1000 dilution of Promega's Anti-ACTIVE® JNK pAb. (0002381)

4. Paumelle, R., Tulasne, D., Leroy, C., Coll, J., Vandenbunder, B., and Fafeur, V. (2000) Sequential activation of ERK and repression of JNK by scatter factor/hepatocyte growth factor in madin-darby canine kidney epithelial cells. *Mol. Biol. Cell.* **11**, 3751-3763.

   Notes: The authors characterize the cell signaling pathways triggered by the multifunctional growth factor scatter factor/hepatocyte growth factor. In Madin-Darby Canine Kidney epithelial cells SF/HGF induces phosphorylation of MAPK while stimulating weakly and then repressing phosphorylation of JNK. The Anti-ACTIVE® MAPK pAb and Anti-ACTIVE® JNKpAb were used to quantitate the level of activation of the MAPK and JNK signaling pathways by Western blot analysis. (0002379)

5. Terstegen, L., Gatsios, P., Bode, J.G., Schaper, F., Heinrich, P.C., and Graeve, L. (2000) The inhibition of interleukin-6-dependent STAT activation by mitogen-activated protein kinases depends on tyrosine 759 in the cytoplasmic tail of glycoprotein 130. *J. Biol. Chem.* **275**, 18810-18817.

   Notes: The effect of suppressor of cytokine signaling (SOCS) expression on the Jak/STAT, MAPK, JNK, and p38 signaling pathways was examined in HepG2, Cos-7, and NIH 3T3 cells. Both PMA and bFGF (Promega) resulted in a rapid upregulation of SOCS-3 expression. MAPK, JNK, and p38 activation was monitored by Western blot analysis using the Anti-ACTIVE® MAPK Anti-ACTIVE® JNK pAb, and the Anti-ACTIVE® p38 pAb. Total levels of active and inactive MAPK protein was determined using the Anti-ERK 1/2 pAb, Rabbit. (0002380)

6. Burns, K., Clatworthy, J., Martin, L., Martinon, F., Plumpton, C., Maschera, B., Lewis, A., Ray, K., Tschopp, J., and Volpe, F. (2000) Tollip, a new component of the IL-1RI pathway, links IRAK to the IL-1 receptor. *Nat. Cell Biol.* **2**, 346-51.

   Notes: Activation of JNK in response to interleukin-1 treatment of 293 cells transfected with

1: Curr Opin Cell Biol. 1997 Apr;9(2):205-12. Related Articles, Links

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T cell antigen receptor signal transduction.

Qian D, Weiss A.

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The T cell antigen receptor (TCR) initiates signal transduction by activating multiple cytoplasmic protein tyrosine kinases (PTKs). Considerable progress in the field of TCR signal transduction has been made in three areas recently: first, in understanding the structure and function of the PTK ZAP-70; second, in the elucidation of the function of the substrates and pathways downstream of the PTKs; and third, in the identification of molecules that negatively regulate TCR signalling.

Publication Types:
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ALX-270-204 **bpV(phen)** [ALEXIS]

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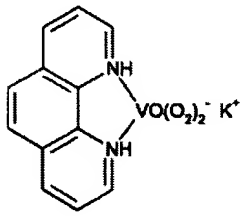

General:

Product-ID	ALX-270-204
Product Name	bpV(phen)
Synonyms	Potassium bisperoxo (1,10-phenanthroline) oxovanadate (V)

Product Specifications:

	$K[VO(O_2)_2C_{12}H_8N_2] \cdot 3H_2O$
MW:	350.3 . 54.1
Purity:	≥95%.
Appearance:	Dark yellow powder.
Solubility:	Soluble in water.
Long Term Storage:	+4°C
Handling:	Protect from light.

Product description:

Potent protein phosphotyrosine phosphatase inhibitor. Also shown to be a potent insulin receptor kinase activator; excellent insulin mimetic *in vitro* and *in vivo*.

Product Specific Literature References:

Peroxovanadium compounds. A new class of potent phosphotyrosine phosphatase inhibitors which are insulin mimetics: B.I. Posner, et al.; J. Biol. Chem. **269**, 4596 (1994) Abstract
Selective activation of the rat hepatic endosomal insulin receptor kinase. Role for the endosome in insulin signaling: A.P. Bevan, et al.; J. Biol. Chem. **270**, 10784 (1995) Abstract
In vivo insulin mimetic effects of pV compounds: role for tissue targeting in determining potency: A.P. Bevan, et al.; Am. J. Physiol. **268**, E60 (1995) Abstract
Hypoglycemic effects of peroxovanadium compounds in Sprague-Dawley and diabetic BB rats: J.F. Yale, et al.; Diabetes **44**, 1274 (1995) Abstract
A role for tyrosine phosphorylation in both activation and inhibition of the insulin receptor tyrosine kinase in vivo: P.G. Drake, et al.; Endocrinology **137**, 4960 (1996) Abstract
Phosphatidylinositol 3'-kinase and p70s6k are required for insulin but not bisperoxovanadium 1,10-phenanthroline (bpV(phen)) inhibition of insulin-like growth factor binding protein gene expression. Evidence for MEK-independent activation of mitogen-activ: C.J. Band & B.I. Posner; J. Biol. Chem. **272**, 138 (1997) Abstract

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